

Neurological presentations of hypothyroidism: the importance of slow relaxing reflexes

M P Wise MRCP FRCP S Blunt PhD MRCP
R J M Lane MD FRCP

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Neurological sequelae of hypothyroidism are well recognized and relatively common. Neurological symptoms occurring as a presenting feature of this illness are, however, uncommon. Here we report three patients who presented in such a way and in whom the diagnosis was only suspected because of slow relaxing reflexes.

CASE REPORT

Case 1

A 24-year-old Asian male computer engineer presented in August 1992 with a 4-5 month history of shock-like pains from the soles of his feet to mid-thorax whilst walking. Over a similar period there had been a progressive slurring and slowing of speech without difficulty in swallowing, and dry, burning or gritty eyes, but without joint or skin involvement. He had also noticed a decrease in mental alertness, but none the less had continued in full-time employment. Diurnal rhythm was altered with insomnia, early morning awakening and excessive daytime somnolence. This sleep pattern had been present for approximately 2 years.

General examination was normal. Extra ocular movements revealed broken pursuit but no frank nystagmus. He had a mild lingual dysarthria and slightly clumsy tongue movements but cranial nerve examination was otherwise unremarkable. Minimal proximal weakness was observed in all four limbs. All modalities of sensation were intact. In the upper limbs he had bilateral

dysidiadokinaesia but a normal finger nose test. The lower limbs showed minimal heel/shin inco-ordination and a little unsteadiness of tandem gait. Reflexes were generally brisk but with a markedly slow relaxation phase. Plantars were flexor. Mini mental state examination produced a score of 24/30.

Full blood count was normal with erythrocyte sedimentation rate (ESR) 3. B12 was low at 100 (160-800) with normal red cell and serum folate levels. Creatine kinase (CK) was markedly elevated at 683 U/L (normal <220). However, the most striking biochemical abnormalities were the total thyroxine which was markedly reduced at <25 nmol/l with an increased thyroid stimulatory hormone (TSH) of 312 mU/l. Fluorescent anti-nuclear antibody (FANA), Rh factor, anti-Ro/La/Sm, anti-parietal, anti-intrinsic factor and thyroglobulin antibodies were all undetectable. Thyroid microsomal antibody titres were markedly elevated at 1/25 600.

Electrocardiogram showed a sinus bradycardia of 50/min with normal complexes. Chest radiograph and echocardiogram were normal as were brain magnetic resonance imaging and myelogram. Electrophysiology revealed no evidence of carpal tunnel syndrome, myopathy or neuropathy. An electroencephalogram was of low voltage but otherwise within normal limits. Cerebrospinal fluid examination showed a raised protein of 1.27 g/L with a normal glucose and no cells.

Treatment with thyroxine was commenced and within 2 months the shock-like pains and eye symptoms resolved. The dysarthria greatly improved and he noticed slimming of his face, restoration of memory, concentration and appetite. Proximal weakness was no longer present (CK normal) and the performance on mental state examination improved to 30/30. However, all reflexes remained slow relaxing.

Now much improved, the patient remarked he had noticed constipation, cold intolerance and general psychomotor retardation prior to initial consultation, although these symptoms had been denied when specifically sought at presentation. On further follow-up his neurological examination returned to normal.

Case 2

A 41-year-old female chartered accountant presented, 1 month after returning from holiday in Antigua. During her visit she sustained a cut in her toe requiring stitches and prophylactic antibiotics. She had subsequently felt non-specifically unwell with myalgia and lethargy during the course of treatment but this later improved. On returning to the UK she once again felt unwell, predominately with myalgia and weakness in her calf muscles. Her nanny, who had accompanied her on this trip, had by this stage

Table 1. Neurological involvement in hypothyroidism

Poor concentration and memory
Lethargy, apathy and drowsiness
Headaches, tinnitus and hearing loss
Decreased psychomotor activity
Dysarthria and hoarseness
Muscle and joint pain, cramps or stiffness
Muscular weakness
Nerve entrapment syndromes
Peripheral neuropathy
Paraesthesia and lancinating pains
Cold intolerance
Incoordination and ataxia
Dementia
Convulsions and coma

developed hepatitis A. Routine investigations showed a normal full blood count and ESR, mild hyponatraemia and a slightly raised alanine aminotransferase of 50 U/l (0–30).

At this junction she was referred for a neurological opinion with a working diagnosis of a 'post-infective myasthenic-like syndrome'. In addition to the symptoms described above, she had noticed mild arthralgia, some swelling of her hands, muscle cramps, a feeling that her face was swollen, slurring of her speech and gross fatigueability. Despite these symptoms she had been working out in a gym without any improvement in her general condition.

On examination the patient had a sinus bradycardia of 54. Mild slurring dysarthria was evident but the cranial nerves were otherwise normal. In the limbs, a mild proximal weakness could be demonstrated. Deep tendon reflexes showed a marked delay in relaxation.

This latter finding prompted further direct questioning at which point the patient admitted her weight had increased and that she tended to feel excessively cold. She had been sleeping poorly and had noticed a recent fall-off in concentration and memory.

Her repeat blood count showed a mild macrocytic anaemia (haemoglobin 11.9, mean corpuscular volume 97), a moderate increase in transaminases, grossly elevated CK (1192 U/l, normal 25–170) and cholesterol of 9.67 mmol/l (normal 5.7). Biochemically the patient was confirmed to be hypothyroid with a free T4 of 5.5 pmol/l and a TSH of >100 mU/l. The patient placed on daily thyroid replacement therapy, with subsequent resolution of all symptoms and neurological signs.

Case 3

A 73-year-old wife of a retired doctor was referred as a newly diagnosed case of Parkinson's disease. Fourteen years

previously she had suffered a subarachnoid haemorrhage resulting in a right-sided hemiplegia and subsequent thalamic pain syndrome. She had recovered well, but over a 6–8 month period had noticed a progressive deterioration in her walking, with increasing clumsiness and dysarthria and a progressive tendency to lean towards the left side.

On examination the patient was rather pale, with sparse hair and waxy skin. She appeared to have a scanning dysarthria with slow tongue movements. The cranial nerves were otherwise intact. No discernable deficit of power or sensation was detected at the limbs. Reflexes were normal in terms of briskness except at the ankle where they were depressed, but there was a striking delay in relaxation particularly at the ankle. Coordination was rather slow and inaccurate but symmetric. Gait was bizarre with marked postural instability.

A full blood count showed a mild macrocytic anaemia. Urea and electrolytes were normal but total CK was grossly elevated at 2040 U/l, with free T4 3 pmol/l and TSH 91.4 mU/l. She was felt to be exhibiting neurological manifestations of hypothyroidism, superimposed on her long-standing but minimal neurological deficits. Following treatment with L-thyroxine, she showed substantial improvement in general well being and her incoordination and gait disturbance improved substantially.

DISCUSSION

Neurological manifestations of hypothyroidism, as outlined in Table 1, are well described¹. However, such problems typically occur in patients with other features of hypothyroidism and it is unusual for this illness to present with predominantly or exclusively neurological symptoms. Our three cases exhibited a syndrome of slurring dysarthria, proximal weakness with a raised CK, and slow relaxing reflexes.

Although patients 1 and 2 had symptoms which in retrospect suggested a diagnosis of hypothyroidism, these became apparent only after treatment with thyroxine, despite specific enquiry at presentation. Myxoedematous patients are often poor historians with few spontaneous complaints², making clinical diagnosis more difficult. The principal complaints in our first patient were shock-like pains, slurred speech, dry eyes and an altered sleep pattern. Poor performance on mental state examination and the recording of a low voltage electroencephalograph suggested a degree of psychomotor retardation but despite this, the patient was able to continue working as a computer engineer. The neurological complaints in the second patient were more suggestive of a primary myopathy, a neuromuscular junction disorder or a post-infectious fatigue state.

Cerebellar ataxia is a well-recognized feature of myxoedema. Certainly our first patient exhibited mild

dysdiadokinaesia, impaired tandem gait and dysarthria, and the third patient had problems with gait and coordination, but these were difficult to interpret in view of the earlier neurological damage. Patients have been described who are mentally alert but in whom ataxia has been prominent¹ with resolution on treatment, but cerebellar atrophy and degeneration have been reported in ataxic myxoedematous patients³.

A striking feature of these patients was the discrepancy between the severity of the biochemical abnormality and the clinical state. The first two patients had continued to work in spite of poor concentration and memory. Similarly, proximal weakness and a raised CK were observed but without abnormality of nerve conduction or electromyography.

The critical examination finding in these patients which raised the possibility of an underactive thyroid, was the observation of slow relaxing reflexes. This finding is often considered pathognomonic of hypothyroidism, although prolongation also occurs in diabetes, ageing and the

puerperium⁴. Generally, there is a poor correlation between the duration of the ankle jerk and thyroid function⁵. However, in the cases described it was the single most important feature amongst a somewhat confusing constellation of symptoms which led to the diagnosis of hypothyroidism; an incapacitating but treatable illness.

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